REMARKS

Claims 1-3 are currently pending in this application. Claim 1 has been amended. Support for the language "secretory" can be found on page 9, paragraph 1, third sentence *et seq.* No new matter has been added. In view of these amendments and of the following remarks, Applicants believe that all the asserted rejections are in condition for withdrawal and all the claims are in condition for allowance.

The Examiner has objected to claim 1, stating that the term "and" in line 2 should be canceled and replaced with the term "or." Claim 1 has been amended as suggested by the Examiner.

REJECTIONS UNDER 35 U.S.C. § 103

Claims 1 and 3 stand rejected under 35 U.S.C. 103(a) for purported obviousness in view of Cheng et al. The Examiner asserts that Cheng et al. disclose treating disorders such as Alzheimer's disease by administering to a patient a p38 MAP kinase inhibitor in a dosage range of a 0.1-50 mg/kg/body weight/day, preferably 0.5-20 mg/kg/body weight/day, and that this range overlaps with and therefore suggests the claimed range of 0.1 ng to 10 mg/kg/body weight/day recited in claim 1.

Claim 1 has been amended to recite administering a *secretory* phospholipase A_2 (PLA_2) inhibitor, which a skilled artisan would know is not an identical active agent to the MAPK inhibitors disclosed by Cheng et al, either *de facto*, inherently or mechanistically, for the following reasons. It is known that arachidonic acid (AA) release and production of eicosanoids are prerequisites for inflammation, and enzymes generally referred to as phospholipase A_2 (PLA_2) are the key enzymes that initiate the AA cascade that results in inflammation. PLA_2 activity can be subdivided into two groups based on structure and enzymatic characteristics: secretory PLA_2 and cytosolic PLA_2. Cytosolic PLA_2 has multiple phosphorylation sites, among which the mitogen-activated protein kinase (MAPK)-directed site is the most critical for its activation. Therefore, inhibition of MAPK with a MAPK inhibitor inhibits only cytosolic PLA_2 activity, not secretory PLA_2 activity. Because both secretory PLA_2 and cytosolic PLA_2

independently are capable of initiating the AA cascade, the inhibition of cytosolic PLA_2 will not inhibit the AA cascade initiated by secretory PLA_2.

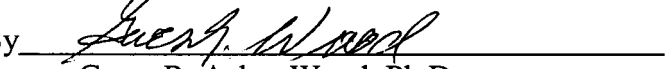
CONCLUSION

Applicants submit that the new and unexpected finding of the claimed invention is neither taught nor suggested by Cheng et al., namely, which inheres in the critical inventive feature that beta-amyloid-induced vasoactivity surprisingly is modified in patients afflicted with Alzheimer's disease or other vascular-related diseases or disorders by antagonizing secretory phospholipase A_2 by administering a soluble phospholipase A_2 inhibitor.

Based on the foregoing amendments and remarks, claims 1-3 are patentable over the cited prior art and in condition for allowance. Reconsideration of the rejections and allowance of pending claims 1-3 are respectfully requested.

Respectfully submitted,

THE WEBB LAW FIRM

By 

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